Amendments to the Drawings:

The attached sheet of drawings includes changes to Fig. 17. This sheet, which includes Fig. 17 replaces the original sheet including Fig. 17.

Attachment: Figure 17 Replacement Sheet
Figure 17 Annotated Sheet Showing Changes

REMARKS/ARGUMENTS

Status of the Claims

Upon entry of the present amendment, claims 26, 35-37, and 52-69 are pending. Claims 59-67 are withdrawn as drawn to a non-elected invention, to be rejoined for examination upon a finding a linking or generic claim allowable. Claims 26 and 58 are amended.

Claim 26 is amended to set forth in the body of the claim that the immune cell deficiency results from the deficient glycosyltransferase activity.

Claim 58 is amended to properly depend from claim 57.

No new matter is added by the present amendments, and the Examiner is respectfully requested to enter them.

Request for Rejoinder under M.P.E.P. § 821.04

Claims 59-67 were withdrawn from examination as being drawn to a non-elected invention, to be examined upon determination of an allowable linking claim. Upon entry of the present amendments, Applicants believe that linking claim 26 is allowable. Accordingly, pursuant to M.P.E.P. § 821.04, Applicants respectfully request withdrawal of the restriction requirement, and examination of the withdrawn claims.

Amendments to the Drawings

The Examiner has requested that Figure 17 be amended to comport with the claims. In response, Applicants have amended Figure 17 to set forth that binding of "Diagnostic Reagent 1" to a sample indicates that the mammal from which the sample was obtained has a glycosylation disorder. Applicants have further amended Figure 17 to set forth that binding of "Diagnostic Reagent 2" to a sample indicates that the mammal from which the sample was obtained does not have a glycosylation disorder.

Amendments to the Specification

The Examiner has requested that the figure legend to Figure 17 at page 11, lines 5-15 and the paragraphs on page 16, lines 5-27 be amended to correspond to the amended Figure 17. In response, Applicants have amended the figure legend to Figure 17 on page 11, lines 5-15 and the paragraphs on page 16, lines 5-27 to correspond to amended Figure 17.

Rejection under 35 U.S.C. §112, second paragraph

Claim 58 is rejected under 35 U.S.C. § 112, second paragraph for alleged indefiniteness. In response, Applicants have amended claim 58 to properly depend from claim 57, not in acquiescence to the Examiner, but solely in the interest of furthering prosecution.

Rejection under 35 U.S.C. § 102(a/b) over Ellies, Immunity (1998) 9:881 ("Ellies")

The Examiner has rejected claims 26, 35-37, 52-58 and 68-69 under 35 U.S.C. § 102(a/b) as allegedly anticipated by Ellies. The Examiner is assuming that the publication date of Ellies predates the filing date of the priority application 60/113,680, filed on December 21, 1998. *See*, page 4, of the present Official Action.

This rejection is respectfully traversed because Ellies is not prior art. Applicants attach to this response an email from Kerry M. Evans, Senior Managing Editor, Current Biology and Immunity, of Cell Press.[1] Ms. Evans affirms that Ellies was officially published on December 22, 1998, one day after the December 21, 1998 filing date of the 60/113,680 priority application. Example 2 on pages 33-44 and Figures 8-13 (including legends) of the 60/113,680 application disclose the same information that was published in Ellies. Accordingly, the Examiner is respectfully requested to withdraw this rejection.

[1] Attached as Exhibit A.

Rejection under 35 U.S.C. § 102(b) over U.S. Patent No. 5,320,943 ("Fukuda")

The Examiner has rejected claims 26, 53-55, 58 and 68-69 as allegedly anticipated by Fukuda. To the extent that the present rejection applies to the amended claims, this rejection is respectfully traversed.

Proper anticipation requires that the cited reference teach or suggest each and every element of the rejected claims.

Here, amended claim 26 sets forth in the body of the claim that the deficiency in immune cell function results from a mutation in a glycosyltransferase gene. Therefore, the phrase "the deficiency in immune cell function resulting from a mutation in a glycosyltransferase gene" should be given patentable weight. A mutation in a glycosyltransferase gene will result in decreased or eliminated expression or activity of the encoded glycosyltransferase. In contrast, Fukuda discloses detecting dysfunction or malignancy in T cell function by detecting alterations of the glycosylation of cell surface leukosialin due to the stimulation or increased activity or amount of β1→6GlcNAc-transferase (Core 2 GlcNAc-T). *See*, column 2, lines 39-42; column 5, lines 52-61; column 6, line 53 through column 7, line 7; Table IV (column 19, lines 1-15) and claims 1 and 10. Fukuda does not disclose the reasons for the increased activity of Core 2 GlcNAc-T. At the time of filing of the present invention, it was known by those of skill in the art that Wiscott-Aldrich Syndrome is due to a defect in the gene encoding Wiscott-Aldrich Syndrome Protein (WASP). WASP was understood by those of skill to regulate the actin cytoskeleton.[2] WASP is not a glycosyltransferase. Fukuda does not teach or suggest detecting any deficiency in immune cell function resulting from a mutation in a glycosyltransferase gene. Whereas deficiencies in immune cell function resulting from a mutation in a glycosyltransferase gene can be detected in any sample from an individual, Fukuda discloses only detecting differences in the glycosylation of leukosialin on peripheral blood lymphocytes, particularly T lymphocytes.

Because Fukuda does not teach or suggest each and every element of the present invention, Fukuda does not anticipate the present methods. Accordingly, the Examiner is respectfully requested to withdraw this rejection.

CONCLUSION

In view of the foregoing, Applicants believe all claims now pending in this Application are in condition for allowance. The issuance of a formal Notice of Allowance at an early date is respectfully requested.

If the Examiner believes a telephone conference would expedite prosecution of this application, please telephone the undersigned at 415-576-0200.

Respectfully submitted,

Jennifer L. Wahlsten
Reg. No. 46,226

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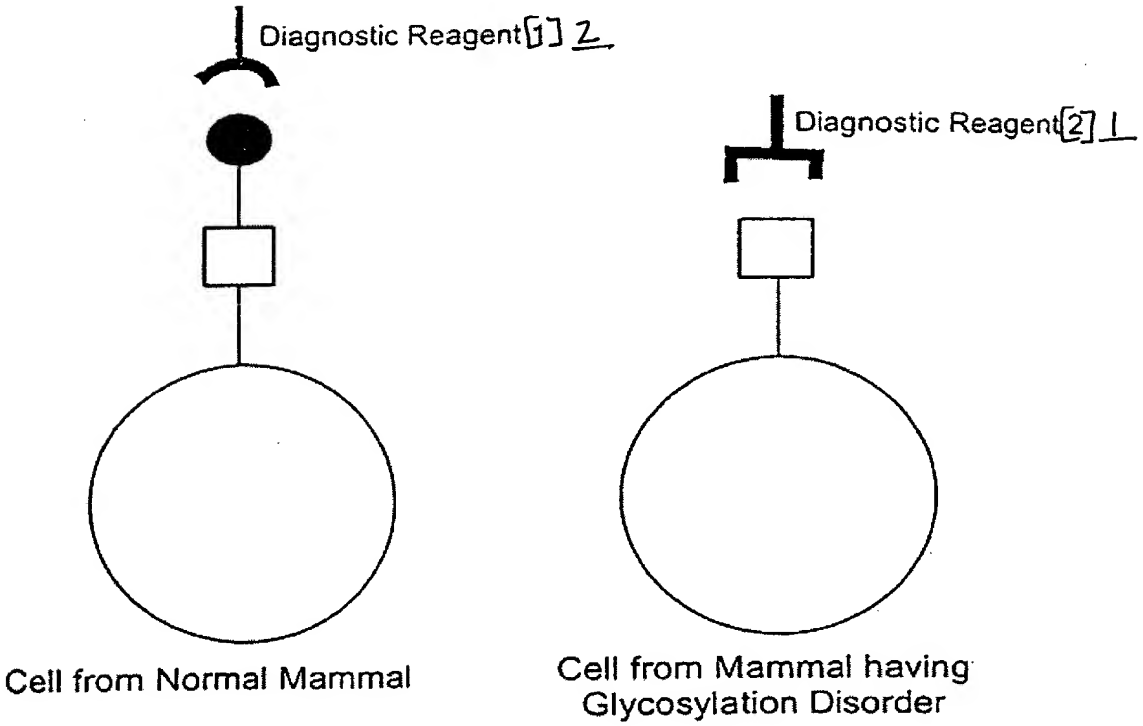
Attachments
JLW:jlw
60845747 v1

[2] Abstract of Symons, *et al*, *Cell* (1996) 84(5):723-34 is attached as Exhibit B.

Figure 17



	Binding of Diagnostic Reagent	
	Normal	Glycosylation Disorder
	+	-
	-	+

Wahlsten, Jennifer L.

From: Evans, Kerry (ELS-CMA) [kevans@cell.com]
Sent: Wednesday, August 16, 2006 9:58 AM
To: Barker, Anne
Subject: RE: Date of Publication question

That is correct.

Best,
Kerry

From: Barker, Anne [mailto:abarker@townsend.com]
Sent: Wednesday, August 16, 2006 12:53 PM
To: Evans, Kerry (ELS-CMA)
Subject: RE: Date of Publication question

Dear Ms. Evans,
Thank you very much for your prompt reply. Can you please confirm that a pre-release copy of the article was not available on-line prior to the publication date?

Thank you for your time,
Anne
Assistant Librarian

-----Original Message-----
From: Evans, Kerry (ELS-CMA) [mailto:kevans@cell.com]
Sent: Wednesday, August 16, 2006 6:00 AM
To: Barker, Anne
Cc: Lee, Peter (ELS-CMA)
Subject: RE: Date of Publication question

Dear Ms. Barker,
The publication date for the below article was December 22 1998.

Please let me know if I can be of further assistance.

Best wishes,
Kerry
****
Kerry M. Evans
Senior Managing Editor
Current Biology and Immunity
Cell Press
600 Technology Square, 5th floor
Cambridge, MA 02139
Phone: 857-998-2249; Fax: 617-397-2820
E-mail: kevans@cell.com
****

Exhibit A

From: Barker, Anne [mailto:abarker@townsend.com]
Sent: Tuesday, August 15, 2006 2:42 PM
To: plee@cell.com
Subject: Date of Publication question

Dear Mr. Lee,
I am trying to determine when the following article was first made available to the public. Can you please provide a date of publication for this article and let me know if it was available on-line prior to being printed?

Lesley G Ellies, Shigeru Tsuboi, Bronislawa Petryniak, John B Lowe, Minoru Fukuda, and Jamey D Marth
Core 2 Oligosaccharide Biosynthesis Distinguishes between Selectin Ligands Essential for Leukocyte Homing and Inflammation
Immunity
Volume 9 Issue 6 Page 881-890 - December 1998
Thank you very much,
Anne Barker

Anne N. Barker
Assistant Librarian
Townsend and Townsend and Crew LLP
379 Lytton Avenue
Palo Alto, CA 94301-1431
Phone: 650.326.2400
FAX: 650.326.2422
abarker@townsend.com
www.townsend.com

1: Cell. 1996 Mar 8;84(5):723-34.

Wiskott-Aldrich syndrome protein, a novel effector for the GTPase CDC42Hs, is implicated in actin polymerization.

Symons M, Derry JM, Karlak B, Jiang S, Lemahieu V, Mccormick F, Francke U, Abo A.

Onyx Pharmaceuticals, Richmond, California 94806, USA.

The Rho family of GTPases control diverse biological processes, including cell morphology and mitogenesis. We have identified WASP, the protein that is defective in Wiskott-Aldrich syndrome (WAS), as a novel effector for CDC42Hs, but not for the other Rho family members, Rac and Rho. This interaction is dependent on the presence of the G protein-binding domain. Cellular expression of epitope-tagged WASP produces clusters of WASP that are highly enriched in polymerized actin. This clustering is not observed with a C-terminally deleted WASP and is inhibited by coexpression with dominant negative CDC42Hs-N17, but not with dominant negative forms of Rac or Rho. Thus, WASP provides a novel link between CDC42Hs and the actin cytoskeleton, which suggests a molecular mechanism for many of the cellular abnormalities in WAS. The WASP sequence contains two novel domains that are homologous to other proteins involved in action organization.

PMID: 8625410 [PubMed - indexed for MEDLINE]

Exhibit B